TABLE 1

CHANGE IN FREQUENCY (q) OF A RECESSIVE GENE UNDER DIFFERENT DEGREES OF NEGATIVE ASSORTATIVE MATING

Generation	c				
	0	− 0.25	− 0.50	− 0.75	− 1.00
0	0.50	0.50	0.50	0.50	0.50
1	0.50	0.54	0.58	0.63	0.67
5	0.50	0.65	0.71	0.74	0.75
10	0.50	0.70	0.73	0.74	0.75
∞	0.50	0.75	0.75	0.75	0.75

two genotypes the third generation will occur as 0.5 $Aa + 0.5\ aa$. This represents the equilibrium condition under which A has a frequency of 0.25 and a of 0.75, regardless of their initial values. But since complete negative assortative mating occurs rarely if at all in nature, a more general method of calculating is needed if the effects of such a mating system are to be conveniently assessed.

Let the genotypes AA, Aa, aa have the respective frequencies α, β, γ; let the alleles A, a have respective frequencies p, q; let the phenotypes $A-$, aa have respective frequencies D $(= \alpha + \beta)$ and R $(= \gamma)$; and let the mating types $A- \times A-$, $A- \times aa$, $aa \times aa$, have the respective frequencies x, y, z $(\alpha + \beta + \gamma = p + q = D + R = x + y + z = 1)$. Finally, let the coefficient of assortative mating c be measured on the usual correlation scale. Since panmixis occurs at $c = 0$ and since only negative values of c are of interest here, we may set up the definition

$$c = \frac{x_- - x_0}{x_0} = \frac{y_- - y_0}{y_0 - 1} = \frac{z_- - z_0}{z_0}$$

where the − subscript indicates negative assortative mating and the 0 subscript indicates panmixis.

Then, with $c = 0$,

$$x = x_0, \qquad y = y_0, \qquad z = z_0$$

and

$$(\alpha + \beta + \gamma)^2 = D^2 + 2DR + R^2 = \alpha' + \beta' + \gamma' = 1$$

where primes indicate the subsequent generation. Collecting the indicated segregation classes according to genotype, gives

$$\alpha' = p^2, \qquad \beta' = 2pq, \qquad \gamma' = q^2. \tag{1}$$

And if gene frequencies remain constant,

$$\alpha' = \alpha, \qquad \beta' = \beta, \qquad \gamma' = \gamma.$$

Similarly, with $-1 \leqslant c \leqslant 0$,

$$x = x_- = x_0 + cx_0 = (1 + c)x_0, \qquad y = y_- = y_0 - c(x_0 + z_0) = (1 + c)y_0 - c, \qquad z = z_- = z_0 + cz_0 = (1 + c)z_0$$

and

$$(\alpha + \beta + \gamma)^2 = (1 + c)D^2 + 2DR(1 + c - c/y_0) + (1 + c)R^2 = \alpha' + \beta' + \gamma' = 1.$$

Proceeding as for (1),

$$\alpha' = (1 + c)p^2, \qquad \beta' = [2q(1 + c) - c/D]p, \qquad \gamma' = (1 + c)q^2 - c\beta/2D. \tag{2}$$

But when $-1 \leqslant c < 0$,

$$\alpha' \neq \alpha, \qquad \beta' \neq \beta, \qquad \gamma' \neq \gamma.$$

When $c = 0$, (2) reduces to (1).

Genotype frequencies for any desired generation may be calculated by (2), used iteratively. Then, since $q = 0.5\beta + \gamma$, under any mating system, the effect of negative assortative mating upon q may readily be determined (Table 1). The determination of the limiting values of q (Table 1), together with the equilibrium properties of negative assortative mating, will be discussed elsewhere. From the present discussion it is clear that the mating system in question necessarily results in changed gene frequencies and that no selective action, as it is customarily defined, is at all involved in the change.

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Weather Influence in Blue Comb in Chickens[1]

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Blue comb in poultry is a pathological complex resembling shock in the early phase, with enteritis and nephritis in less acute cases. Accumulated data on field outbreaks in Florida indicated that climatic stress might be a contributing factor in the development of the disease. A maximum temperature exceeding 85° F was found to occur 3 or 4 days before cases were presented to the laboratory for diagnosis. Cold weather stress was an insignificant factor in the development of outbreaks, probably on account of the low humidity.

The possibility that the climatic stress was a local factor encouraged the study of climatic stress in another weather province. The New England states were selected because they have had experience with blue comb. Several diagnostic laboratories are available, and geographic variability is less than in many other service areas.

During 1951, the maximum temperature recorded at Boston was used to provide the state diagnostic laboratories with a prediction of blue comb. They were asked to report the days when cases were presented to the laboratory. Several factors enter into this comparison. A less observant poultryman might delay presenting the problem to the laboratory. Some cases of blue comb not due entirely to climatic factors may

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TABLE 1

Predicted and Actual Diagnosis of Blue Comb in 1952*

Predicted		Mass. Amherst	Mass. Waltham	Conn.	N. H.	R. I.
Jun	6–12		4, 10, 13	3		
	15–20	20, 23, 23	17, 23	23	20	
	28–30	30	25, 25, 26, 30		30	
Jul	3–7	8, 9	3, 7, 8, 10, 10			
	13–20	14	14, 14, 14, 15		15, 15, 16	
	24–30	29, 30	30		17, 25, 28	
Aug	6	7, 8	6, 8, 11			
	12–19	19		12, 15, 21, 21	13	23
Sep	3	2	3, 3, 4, 5	2		
	11–14	9, 12	16			11
	20	24, 26, 26	22, 26	19, 22, 23		
	29	29				
Oct	7	1, 10	2			
	16	22	14	16, 20		
	29		28, 30			

* Dates in first column represent days when diagnoses were predicted. Dates in other columns represent outbreaks diagnosed.

accompany leukosis or Newcastle disease, or heavy egg production. The pathologist might separate the acute and the subacute phase, or use a different nomenclature.

The result of the survey was promising, as the five laboratories received 71 cases or in within 2 days where temperature had been 78° F or higher. Of 31 other cases, 6 occurred during January to April, and 9 from October to December. Prediction accuracy was not good in 1951, as it was thought that fowl might become acclimatized to hot weather in July and August.

In 1952 a similar prediction was made. Prediction accuracy was better during the hot months, but the influence of cold weather stress needs further review. The results of the prediction for June to October (Table 1) serve to encourage the further study of climatic influence on the development of blue comb in poultry.

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The Effect of a Noxious Stimulus in Man on the Antidiuretic Activity of the Blood

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In both man and dog, exposure to a noxious stimulus results in a marked inhibition of the diuresis induced by the ingestion of a water load (*1*). This antidiuretic response to a noxious stimulus is diminished in the absence of the neurohypophysis (*2*). Consequently, it has been postulated that the antidiuretic response is dependent upon the secretion of an antidiuretic hormone by the neurohypophysis. Direct evidence of such secretion into the circulation is not available.

With the development of a relatively simple, sensitive, and precise procedure for the assay of antidiuretic substances (ADS) in the blood plasma (*3*) it became possible to determine directly the response of the blood ADS to a noxious stimulus.

Kelsall has demonstrated that a short period of ischemic pain in the arm may inhibit the diuresis produced by a water load in man (*4*). Accordingly, healthy men were given 1000 ml of tap water and the urine voided every 10 min was replaced with an equal volume of water in order to maintain a constant water load. When the rate of urine excretion exceeded a volume of 10 ml/min for 2 consecutive periods, a venous blood sample was drawn. Shortly thereafter, the circulation through the right arm was occluded by a sphygmoma-

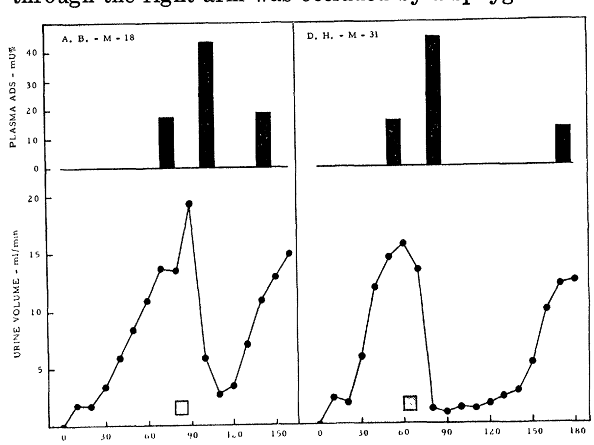

Fig. 1. The effect of a noxious stimulus on diuresis and on the antidiuretic activity of blood plasma. Ischemic pain of an arm was produced during the period marked by the shaded area.

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